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Case Report

Rehabilitation Benefits in Young Patient with Stroke After Foramen Ovale Patent

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Abstract

According to the latest medical literature, approximately 40% of ischemic strokes do not have an identifiable etiology and are called cryptogenic strokes [1]. Patent oval foramen, defective closure of the interatrial septum, may be considered a risk factor for cerebral embolism [2]. In this material, we present the case of a 22-year-old man diagnosed with secondary ischemic stroke foramen ovale patent to be presented for endovascular closure of the septum. The purpose of this presentation is to present the relevant clinical manifestations of the patent oval foramen and cryptogenic stroke, the clinical manifestations, the diagnostic stages, the management, and the therapeutic possibilities of the medical recovery program.

Keywords: *Interatrial Septal Abnormalities; Paradoxical Embolism; Cryptogenic Stroke*

Introduction

The incidence of stroke in Europe in the last years was 1.12 million cases per 12 months and almost 40% of them don't have a certain etiology [1,3]. Cryptogenic strokes have a high incidence in population, especially among young patients [4]. We corroborated the investigations performed with our patient and reviewed also the clinical and paraclinical investigations mentioned in literature in this type of pathology and we must note the high prevalence of defective closure of the interatrial septum such as foramen ovale patent (PFO) [5]. Patients with PFO have no representative symptoms, some of them cause headaches, but these pathology associated with minimal embolic alterations represent the mechanism that produces cryptogenic strokes [5]. In order to reduce the risks produces by PFO, medical literature mention the importance of introducing a screening method for foramen ovale patent cases among general population, especially for young people accusing headaches [5-7].

Treatment of foramen ovale patent includes medical treatment, such as antithrombotic and PFO percutaneous closure, with better results mentioned in literature in case of transcatheter closure. [8] Rehabilitation treatment protocol in case of stroke with PFO mechanism must take in consideration the stabilization of oxygen arterial saturation and cardiac parameters that can be easily influence by the postural changes [9].

Case Report

We present the case of a 22-year-old patient, with no personal history of pathology, except for sporadic migraine episodes and without further investigation, presented urgently on 12.07. 2021 for a decrease in muscle strength in the left hemibody and sudden asymmetry of the face. The patient denies any injury. He is a student and lives in an rented apartment with his family. Regarding AHC there is no significant pathology to mention and also the patient has no comorbidities and does not drink alcohol or does not smoke.

Objective examination at the time of emergency reveals: constitutionally hyperpigmented skin and mucous membranes, impalpable superficial lymph nodes, normally conformed thorax, MV present bilaterally, without pulmonary rales, SaO_2 98% aa; BP 120/80 mmHG, AV 93 bpm rhythmic, palpable pulses bilaterally, no signs of peritoneal irritation.

Regarding the neurological examination at admission we mention no involuntary movements or signs of meningeal irritation, no visual acuity disorders, left homonymous hemianopsia, preserved oculomotricity, RFM + direct and consensual, central facial left paresis, no swallowing disorders for solids and liquids, tongue on the line median in situ and protrusion, orthostation and gait impossible, left hemiplegia, left lower limb anesthesia, left upper limb hypoaesthesia, euphasia, perform simple and complex orders.

Investigations performed

EKG: Sinus rhythm, 93 bpm AV, intermediate QRS axis, minor BRD, no ST-T segment changes.

Cardiological evaluation - BP 105/60, av 68 bpm, SO_2 98% aa, no resting dyspnea, no audible detec Thrombophilia heart murmurs, no edema/hepatomegaly, symmetrical peripheral pulse. Transesophageal ultrasound: undilated left atrium, no images of in-ear thrombi, good entry and exit speeds (approximately 1 ml). Left undilated ventricle with good kinetics, good systolic function (correlated with TTE = transthoracic ultrasound); Undilated RV, preserved longitudinal function. Slim-looking mitral valve, no images suggestive of infectious endocarditis, no regurgitation with hemodynamic significance. AO valve: tricuspid with a supple appearance, without images suggestive of vegetation, without regurgitation, with good opening V tricuspid/pulmonary with a supple appearance. At the level of the interatrial septum, the presence of patent oval foramen with a passage of the right-left contrast solution is highlighted, at the Valsalva maneuver and also with color Doppler flow present at this level, PFO with dimensions of approximately 12 mm (measurement in bicon incident) G = 3 mm, Eustachian valve with a length of 18 mm, ascending aortic and crosal, without visible atheromatosis.

Ophthalmological evaluation: The patient accuses mobile "blackheads" with eyeball movements, FAO with flat papilla, clear contour, physiological excavation, vessels of normal caliber and appearance, macula with present reflex, without pathological changes. Diagnosis: vitreous disorders. General suppressive treatment recommendations 3 months

Native brain CT 12.07.2021: No recent endocranial changes, ventricular system located on the median edge with normal dimensions, calcified parietal atheromas at the level of bilateral ACI in the intracavernous segment; no collections at the level of the paranasal sinuses; left paramedian posterior fossa arachnoid cyst (approximately 23/28/40 mm). See figure 1.

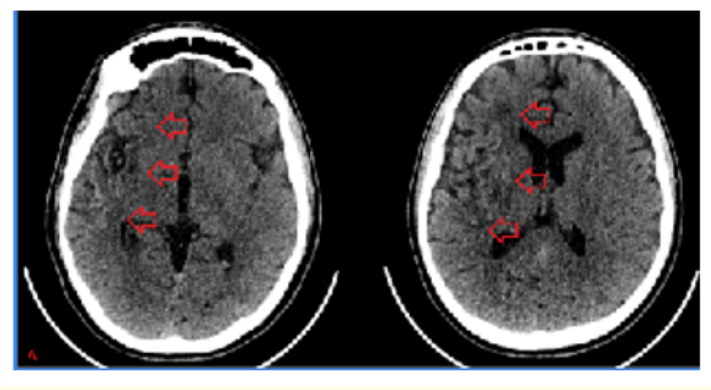

Figure 1: *Native CT. Stroke in right MCA.*

Post-contrast brain CT IV 12.07.2021: Lack of opacification of the intracavernous ACI segment with incomplete homogenization of the right ACM in the M1 segment (successive stenoses); the rest of the main endocranial arterial vascular axes homogeneous postcontrast iv. See figure 2.

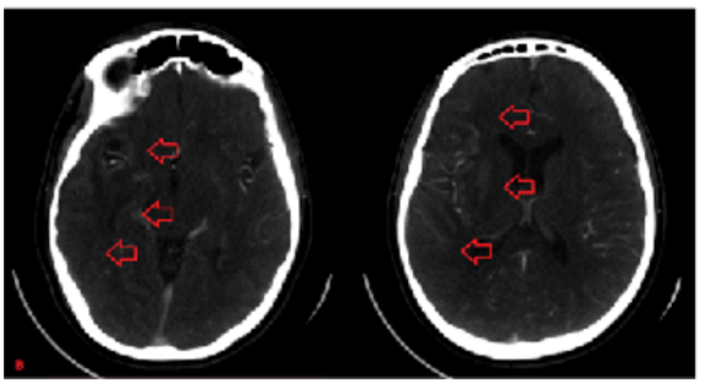

Figure 2: *Post-contrast brain CT IV. Stroke in right MCA.*

IV thrombolysis is performed on presentation on 12.07.2021. NIHSS 15 points at the presentation.

Native brain CT (post thrombolysis) 13.07.2021- Low hypodensity with acute ischemic appearance projected cortico-subcortical frontally on the right side and erasing the white matter-gray matter differentiation at the temporo-insular and capsulo-nuclear level on the same side; Right ACM with spontaneous hyperdensity; midline ventricular system, with straight VL learning; without other notable changes compared to the previous examination.

Cervical MRI with angio MRI (20.07.2021) TOF arterial and dynamic postGd-ACI dr with faster attenuated and narrowed rapid flow (compared to left ACI, cc 55% of ACIstg diameter) on the entire cervical and intracarotid segment, with a similar load as diameter and intensity at dynamic postcontrast angioMRI, but without wall anomalies detected in segments T2 or T1fs pre/post Gd.

AngioCT result supra-aortic trunks: (03.08.2021) brachiocephalic trunk, aa subclavian and bilateral ACC without stenosis; ACIdr permeable, smaller caliber from the post bulbar level to the level of the carotid T; without a suggestive image of ACIstg dissection fold and bilateral AV without stenosis. Endocranial - AM as permeable, slightly smaller caliber than the left, ACoA and ACoP straight permeable (functional Willis polygon); segment A1 as hypoplastic in ½ proximal. N.B. right fronto-temporo-insular cortico-subcortical hypodensity, characteristic of a subacute stroke. See figure 3.

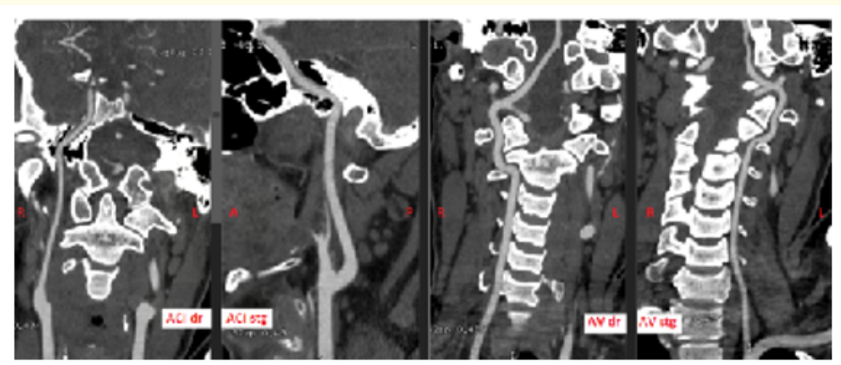

Figure 3: *AngioCT result supra-aortic trunks.*

Rehabilitation programme

On 6.08.2021 the patient was admitted to the Medical Recovery Service accusing mechanical pain in the left shoulder (VAS 7/10) especially in flexion and abduction of the arm, pain in the left hip (VAS 8/10), a motor deficit of plegic intensity in the left hemibody and severe loco motor and self-care deficit.

General objective: good general condition, conscious, cooperating, normal weight (184 cm, 90 kg), normally conformed chest, bilateral symmetrical rib extensions, physiological MV, no rallies, rhythmic heart sounds, no auscultatory detectable murmurs, BP 135/70 mmHg, AV 71 beats/min, bilateral pulsating peripheral arteries.

NMAK left-hemispheric examination: Left central facial paresis, cutaneous hypoaesthesia, left MS: shoulder subluxation, reducible elbow flexum, reducible finger flexum, moderate spastic hemiplegia, muscular hypotrophy, pain in the mobilization of the left joint and shoulder, exhausted clonus, 5 exhaustible clonus tactical, thermal and painful sensitivity disturbance of hypoaesthesia type, CMV PID absent, left MI: motor deficit of plegic intensity, moderate spastic, pain on the mobilization of the left hip, live ROT, clonoids, RCP in extension, CMV PID absent, sensitivity disorder tactical, thermal and painful type of proximal and intermediate hypoaesthesia and distal anesthesia type, inexhaustible clonus, moderate muscle hypotrophy, performs the transfers on the bed alone, keeps the sitting short, moves with a wheelchair. Barthel Index 10/100; ADL 1/10; IADL 1/8; moderate depression on the Hamilton scale.

ECG investigations: Sinus rhythm, axis qrs oriented at 0gr, without repolarization disorders.

RX left shoulder and left hip: No post-traumatic injuries.

Differential diagnosis: Pain in the shoulder joint - impingement syndrome - excluding ultrasound; adhesive capsule - excluded ultrasound; humeral fracture/scapula - radiographically excluded; osteoarthritis of the glenohumeral joint - radiographically excluded.

Of central motor neuron syndrome: Hemorrhagic stroke; Intracranial tumor; CBT - successive CT examinations.

Objectives of the recovery program: Control of associated diseases; Educating the patient and the family about the associated ailments and the involvement of the family in the treatment; Improving motor control (MIstg); Improving transfers and bed mobility; Improving gait pattern; Improving coordination and balance; Improving pain symptoms, increasing exercise tolerance; Prevention of vicious posts and withdrawals.

Treatment: Hygienic diet; Anticoagulant treatment (rivaroxaban); muscle relaxant (baclofen); cerebrolysin; Analgesic (paracetamol 1500 g/day).

Rehabilitation programme

- Electrotherapy techniques: Left shoulder laser-tissue repair, stimulation of fibroblasts, decreases inflammation.
- Tense shoulder stag and left hip 110 HZ 15 min - analgesic.
- US shoulder and left hip 0.6 w/cm^2 5 min - tissue repair (++ mast cells); decontracting, analgesic (micro-massage).
- Left hemibody decontracting sedative massage.
- EMG biofeedback.
- Physical therapy.
- Increasing shoulder mobility - passive, passive-active, active mobilizations.
- Improving motor control - script therapy.

- Decreased spasticity of MS - proprioceptive neurofacilitation techniques (vibrations - tendons, stretching, KAbat diagonals).
- Improving the walking pattern (walking on the field with obstacles, walking on the treadmill at low speed).
- Improving coordination and balance - slight imbalances in sitting position shortened; balance board exercises; Frankel exercises pt MI.
- Increased exercise tolerance - exercise bike, walking on the treadmill.
- Occupational therapy.

The patient followed this physical-kinetic program with good tolerance and slow favorable evolution, a slight improvement of the joint mobility of the shoulder and left hip (Flexion, active abd 60 gr shoulder, and 50 gr hip); slight decrease in pain symptoms; improving the walking pattern - increasing the walking perimeter, increasing the walking speed, improving the balance while walking; Increased exercise tolerance (walking > 30 min with cane support).

NMAK II examination 90 days after the first hospitalization in the Medical Recovery Department MS stag: limitation of active and passive mobility in the shoulder joint: ABD arm 45 degrees; arm flexion 45 degrees; Engine control present P-I-D; Spasticity (MAS) finger flexors gr 2; 2nd-degree pronators; flexori cotg rad 2; ROT accentuated on the left side; Reflex Marinescu Radovici, Hoffman present; asymmetry (hypermetry); MIstg: motor control present proximal, diminished intermediate and distal; Spasticity (MAS) grade 1 knee extensors; plantar adductors and flexors; Left accentuated patellar reflex, positive Babinski. Functional: grip, low strength pliers; mobility in bed and independent transfers, done with difficulty. Walking: support in tetrapodal cane for short distances (approximately 15m), modified scheme, small steps, plantigrade attack left foot, deficient flexion of the knee and left hip, spasticity on Sunday, reduced speed of movement. ADL-B 5/10; ADL-I 4/8; Barthel 50/100; mild depression according to the Hamilton scale.

Antiphospholipid syndrome profile, extended ANOT profile BLOT within normal limits.

C protein	113	70 - 140/%
Lupus Anticoagulant confirm	29.9	27,6 - 36/sec
Antitrombina III	125	83 - 128/%
Lupus Anticoagulant confirm (Lupus C ratia)	1.18	
S protein	114, 7	74.1-146.1/%
FV Leiden - APCRV=	87.6	--/sec
Lupus anticoagulant screen	40.2	29-38.76/sec
FV Leiden - APCRV- I ratia APCR V /FV_Leid(-)sec /HONIOCIS	2.74 32.0 10.4 4	->=2.3 28-184/sec 4.3-11.1/umol/l

Table: *Thrombophilia tests.*

Results and Discussion

The link between Patent Oval Foramen and Stroke is still quite insufficiently studied and defined [1]. Our young patient did not have any other associated condition, except for those migraine-type episodes for which he did not undergo specialized evaluation and treatment, having a rather low intensity and a rare frequency.

Nominal interatrial septum has been identified as an additional risk factor for stroke in young people with stroke [2]. Although the mechanism by which they cause embolism is the reason for much debate in medical sessions, recent studies have shown that Foramen Ovale Patent is often an accidental finding, with a higher prevalence in young stroke patients than [1,2,10-14]. Extensive studies are needed to define the link between interatrial septal abnormalities and stroke in young people.

Several hypotheses are presented in the medical literature that claims that most neurological manifestations are secondary to the paradoxical embolism of small thrombi that occur in the venous system and pass through PFO. However, no direct evidence for cryptogenic stroke and Doppler evaluations for the identification of deep vein thrombosis in the lower limb or pelvis are reported, the results of which are highly variable [12-14].

Other possible causes for PFO secondary stroke, regardless of the embolic phenomenon, are secondary cardiac arrhythmias or endocardial abnormalities in the septum or PFO, which can be identified as sources of risk for thrombus formation [14].

Conclusion

The young patient suffered an acute ischemic stroke, with no history of trauma, no direct signs of AO dissection, there is a clear oval foramen, but no peripheral thrombosis, evidence of thrombophilia within normal limits. The following are undergoing - an antiphospholipid syndrome profile extended ANA profile BLOT. IV thrombolysis was performed on 12.07.2021, NIHSS 15 points on presentation at CG. 2 weeks away NIHSS 10pct. Thrombectomy could not be performed.

Author Contribution

All authors have read and agreed to the published version of the manuscript and have equal contribution in realizing the article.

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Institutional Review Board Statement

All procedures performed in the study were in accordance with the ethical standards of the institution and with the 1964 Helsinki Declaration and its later amendments or compared ethical standards.

Informed Consent Statement

Written informed consent was obtained from the patient in order to publish this paper.

Conflicts of Interest

The authors declare no conflict of interest.

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